AUDITORY LATE LATENCY POTENTIALS IN

LEARING DISABLED CHILDREN

Reg. NO.M9715

Independent Project submitted as part fulfilment for the first

year M.Sc, (Speech and Hearing) to the

University of Mysore.

ALL INDIA INSTITUTE OF SPEECH AND HEARING

MYSORE 570 006
1998

Gurur Brahma
Gurur vishnu:
Gurur Devo Maheswaraha.
Guru Sakshath Parabrahma
Taismai Shree Guruve Namaha

Parents were my first teachers ,and
Teachers my, second parents,
I am indebted to them,
for they have helped me climb
the ladder of knowledge.

This work of mine is dedicated to my
TEACHERS[1].

CERTIFICATE

This is to certify that this Independent Project entitled **AUDITORY LATE LATENCY POTENTIALS IN LEARNING DISABLED CHILDREN** is the bonafide work in part fulfilment for the degree of Master of science (Speech and Hearing) of the student with Register NO.M9715

Mysore
May, 1998

Dr.(Miss) S.Nikam
Director
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CERTIFICATE

This is to certify that this Independent Project entitled *A UDITORY LATE LATENCY POTENTIALS IN LEARNING DISABLED CHILDREN* has been prepared under my supervision and guidance.

Mysore

May, 1998

Lecturer in Audiology
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DECLARATION

This Independent Project entitled.A*UDTTORT LATE LATENCY POTENTIALS IN LEARNING DISABLED CHILDREN* is the result of my own study under the guidance of ***Mrs.*** *C.S.* ***Vanaja,*** Lecturer in Audiology, All India Institute of Speech and Hearing, Mysore and has not been submitted earlier at any University for any other diploma or degree.

Mysore
May, 1998

Reg. NO.M9715

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In the darkness and in light,
In the rain and in storm,
through the darkdays of my life, to the bright ones,
Ma and Papa, its your strength
guidance and hope in me
that has driven me through....
I am stuck with no words to express....
Love you lots, is all what I can say !

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Its your prayers and blessings, that has got me success in journey of life.

* Some one pleasant,
Some one Caring,
Some one Loving,
Some one ....

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Who make this world
a better place tolive in"
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U lived in my hands and
Filled in my thoughts.
My craze for you.
Will never reduce.

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INTRODUCTION

The poetic phrase, *"Words written on water"*, evokes an ephermal and transitory image. Speech is no ephermal and no less transitory. The spoken message is a rapidly decaying acoustic disturbance in the ocean of air. Thus spoken message is always a history and after the acoustic event has passed, a neural image is retained to support its linguistic interpretation; but the same acoustic event cannot be repeated, only similar ones could be obtained. Inter-relations between a hierarchy of levels, auditory, phonetic - phonologic, syntactic and semantic, processing becomes essential for this complex phenomenon of speech comprehension. Thus, the initial stage being *"Auditory perception"*, detailed evaluation of auditory perception and processing in learning disabled becomes essential.

These days, especially, increasing number of children with learning disability are being referred to audiologists for evaluation of possible *"Central Auditory Processing Disorder"*. The process of auditory processing involves, attention, detection and identification of the signal. Thus, any test used for detection of CAPD (Central Auditory Processing Disorder) tests any or all of these stages. Cognitive potentials test "attention" of the individual to detect disruption in cognition and auditory processing.

Definition of Learning Disabled

> "Learning disabled/disability is a general term that refers to a heterogenous group of disorder manifested by significant difficulties in the acquisition and use of listening, speaking, reading, writing, reasoning and/or mathematical abilities. These disorders, are intrinsic to the individual presumed to be due central nervous system dysfunction and may occur across a life span, but mostly it is found as a developmental disorder" (NJCLD, 1988).

Having a Look at its Etiology

Theories about etiology of learning disability, suggest that at least some poor readers, may be distinguished from normal readers, by the peculiarities in their pattern of localisation (e.g., bilateral language

representations), by some dysfunction with in a cerebral hemisphere or by a dysfunction or disturbance in interhemispheric transfer function (Orton, 1928). It is also speculated that the malformations in molecular layer of the left hemisphere may be associated with significant rewiring of the cortex engaged in language (Galaburda, 1989).

Furthermore, it has been hypothesized that learning disabled children show reticular formation malfunctioning (Ayers, 1972; Willefood and Burleigh, 1985). Also, children with learning disability have been characterized as having pervasive deficits in auditory processing and increase in development of auditory skills facilitates reading process.

Thus factors underlying learning disability have been hypothesized to include perceptual and/or intersensory deficits, developmental lag in cerebral dominance, incomplete, mixed or reversed dominance, deficiencies in temporal processing of verbal stimuli and deficiencies in translating visual patterns to verbal codes or vice versa (Vellutino, 1978).

According to Ford, Johnson and Myklebust (1967), auditory visual integration is very significant aspect of learning disability. Evans (1967) draws the following conclusions concerning auditory and audio-visual integration skills as they relate to reading;

(i) in a developmental sense the ability to discriminate auditorily may be important in developing the visualised vocabulary.

(ii) skill in audio-visual and visuo- auditory sensory integrations are positively correlated with reading achievement.

(iii) poor readers appear to be significantly more impaired in these integrative skills than good and normal readers.

(iv) enough evidence appears to exist to warrant attention to auditory functions in preparatory or remedial reading classes.

Evans (1967) further reported that the perceptual problems might occur due to -

(i) Problems in temporal ordering of auditory impulses, incident at the temporal cortex.

(ii) Problems in coding of auditory impulses at the terminal cortical areas of temporal cortex.

(iii) Faculty transmission of impulses along the synaptic junction of the brainstem.

Thus no specific etiology has been established, learning disability could be due to any one or a combination of the above mentioned etiologies.

Auditory Processing in Learning Disabled Children

To-date, the relation between auditory perceptual and language learning abilities has not been clearly established. Measures used to study the perceptual deficits in learning disabled have included both behavioural and electrophysiological tests.

Behavioural Studies and Learning Disability

Behavioural tests like dichotic listening tasks using digits, phonemes, MLD etc. were the ones used initially for studying the relation between cerebral specialization and reading disabilities. These have not led to any clear conclusions. First of all, it is not known whether the tasks and measures used, such as dichotic listening tasks (Bryden, 1970), dichotomous haptic presentations of shapes (Witelson, 1977) are even reliable indicators of cerebral specialization (Shankweiler, Studdert-Kennedy, 1975). Rourke (1978) said that, "... *the relationship between ear advantage and cerebral asymmetry and disabilities in reading remains a mystery".* Also, many of the behavioural studies relating cerebral specialization to reading .. disabilities, have not taken into account task variables, subject selection criteria etc., that influence measures of cerebral specialization (Kinsbourne, 1970, 1973; Heilige and Cox, 1976; Friedman and Poison, 1981).

Electrophysiologic Potentials in Learning Disabled

Electrophysiologic potentials that have been studied include both endogenous and exogenous potentials.

Jerger and Jerger (1981) recorded middle latency response (MLR) and late latency responses on eleven and a half year old learning disabled child using auditory clicks. Their results show that MLR waves had poor morphology than normals, while LLR waves were well defined in both ears of the LD child. Results of study by Jirsa and Clontz (1989, 1990) on LD children showed increased P2-P3 interpeak latency. But found no difference in the LLR amplitudes in LD children. On contrary Pinkerton et al. (1981) found lower Nl amplitudes in learning disabled than normals.

Another study by Arehole (1995) on dyslexics without attention deficit disorder showed longer P2-P1, interpeak latency, but N1-P2 amplitude differences were reported to be very small in comparison with the normals. But all subjects were reported to show good wave morphology.

Also, recently Korpilahti and Lang (1991) reported that Mismatch Negativity (MMN) could be used in testing auditory processing problems. They recorded MMN in 14 learning disabled and found that duration of MMN was shorter than that observed in normals, while latency of MMN was unaffected.

NEED FOR THE STUDY

A review of literature shows that though number of investigators have studied auditory processing in learning disabled children, they have not led to any conclusive results. Also, linguistic behavioural tests using speech material, becomes language specific and thus difficult to use in a multilingual country like India. These give rise to the need for more objective and language nonspecific tests like Event Related Potentials in this field. As these ERPs are direct reflectors of the changes in brain with processing of a stimuli, identifying a particular pattern in learning disabled and comparing it with age matched normal subjects, would help us get a better understanding of the complex procedures of processing of brain in learning disabled children. Thus, there is a need to study all these potentials on the

same subject to get a profile of these in learning disabled children and thus use it in their diagnosis.

AIMS OF THE PRESENT STUDY

(i) Studying the following potentials in learning disabled :

(a) P1,N1,P2,N2

(b) P300(c)MMN

An attempt was made to study

(a) The absolute latency of each wave
(b) Interpeak latency of these waves
(c) N1-P2 amplitude and N2 - P3 amplitude
(d) Duration of MMN.

REVIEW OF LITERATURE

Event Related Potentials (ERP) have provided a means to uncover important aspects of the neural basis of many developmental disorders. Several researchers have found ERP differences between normals and those with autism and attention disorders.

Tracing back in literature, the earliest study of endogenous potentials on learning disabled used visual stimuli. It was Conners et al. (1971) who reported reduced amplitude of Visual Event Related Potentials (VERP) in

the left parietal areas of the lesring disabled population. Early attempts at replication of these findings using VERP, showed inconsistent results. This was mainly attributed to problems in measurement and sophistication of instruments. But later on, with development of tools to measure accurate amplitudes and latencies of evoked potentials, both auditory and visual evoked responses have been recorded in learning disabled children. The results of majority of studies suggest auditory processing problems in learning disabled children.

The Electrophysiological Measures Used in the Present Study are :

Late Latency Exogenous Potentials

PI Nl P2 N2

Late Latency Endogenous Potentials

P300 and . MMN

INTRODUCING THESE POTENTIALS

Auditory Late Latency (Exogenous) response (ALR) are recorded in a time period from about 50 to 250 msec, after the acoustic stimulation at a relatively slow rate (one stimulus every 1 Or 2 secs) (Cited by Hall, 1992). The main components of exogenous potentials are PI (50-80 ms), Nl (100-

150 ms), P2 (150-200 ms) and N2 (180-250ms) (cited by Hall, 1992). The labels for these peaks refer to the expected voltage polarity of the response as recorded from the vertex. ALR was first described by Davis et al. in 1939. They described an *"On Response"* to sound in EEG and used the term *'K-complex'* to describe it (Davis, Davis, Loomis, Harvey and Hobert, 1939).

The changes occurring in brain's electrical activity, in response to internal events such as cognition or perception are referred to as Event Related Potentials (ERPs). ERPs are considered to be endogenous in nature. These occur in proximity to the stimuli, but are relatively invariant to changes in the physical parameters of eliciting stimulus (Desmedt and Debecker, 1979; Donchin et al. 1978). One of the most popular and widely employed ERP is P300. The response is so named because it is a vertex positive wave component occurring from 250 - 600 msec post stimulus. Picton et al. (1977) stated that "… *they are the best evoked potential measurements available, if they can be reproducibly recorded as their presence indicates the complete integrity of the auditory pathway in central nervous system"*.

Infrequent, *"deviant"* stimuli occuring in a sequence of repetitive, *"standard:* sounds elicit a mismatch negativity (MMN) of the auditory event related potential (ERP) (cited by Naatanen, 1990). MMN, usually peaks at 150 to 250 msecs from stimulus onset and overlaps the negative Nl and positive P2 components. MMN appeared to consist of 2 sub components. First a 150 to 200 msec, from stimulus onset and other at 200 to 300 msec (Paavilainen et al. 1991). Thus MMN is elicited when a repetitive sound occasionally changes in frequency, intensity, duration, spatial locus of origin or when a constant time interval between successive stimuli is occasionally shortened. The auditory MMN, appears to provide precise feature specific information about the accuracy of the central sound representation in human brain (cited by Naatanen and Alho, 1997). Importantly for clinical and other applications, MMN can be elicited independently of attention in the sense that even full withdrawal of attention does not abolish MMN. MMN can be interpreted as reflecting a code of stimulus difference or change, that is, in a sense, as a higher order response;

the MMN is difference between two consecutive stimuli. MMN indeed represents a higher order response, one signalling the detection of a difference between two consecutively presented stimuli. Thus MMN, does not represent a code for any stimuli but rather for stimulus change.

ORIGIN

The origin of ALR has been debated for long. Davis (1939) showed that ALR could be recorded from electrodes at numerous scalp location with maximum amplitude from midline electrodes over frontal region. He concluded that the generators were diffuse and non-specific in thalamus and thalamocortical regions. Picton et al. (1974) postulated the possibility of association cortex of frontal lobe to be generators. Evidence has been accumulated that, several concurrent sources contribute to scalp potentials in latency region of ALR (Walpaw and Penry, 1975). But none yet are able to correctly identify the generators of ALR waves.

P300 ERP is supposed to have originated from non-specific unknown neural generators and is felt to be an electrophysiological manifestation of strategies used by CNS in selective attention activities, including frontal cortex, auditory cortex of superior temporal lobe, hippocampus and associated brain sites (Courchesene, 1978; Okade, et al. 1983 Kilany, 1985). Buchwald (1990) has suggested that although the generator sites of P300 are still unknown, the maturation of P300 provides some in sight into the ontogeny of the developing brain for cognitive versus sensory brain systems. Typically the components of ERP appear between 200-600 msec, after the onset of stimuli. These are believed to reflect nonsensory, cognitive processes carried out by human brain (Donchin et al. 1978). Pineda et al. (1989) concluded that nucleus coeruleus plays a major role in *'Modulation'* and *' Generation'* of P300 response based on their study on monkeys. Although the precise generators are still not fully resolved, there is evidence of a subthalamic and medial geniculate origin, with other activity noted in the gyrus orbitalis, rostral thalamus and anterior commissure (Helgren et al. 1980; Wood et al. 1980, 1983). Put into simplest form, the P300 includes responses from the frontal cortex, centeroparietal cortex and hippocampus.

Supratemporal cortical activity is found to have major contribution to MMN elicited by different kinds of stimulus changes. According to Levanen et al. (1992) right hemisphere plays an important role in origin of MMN apart from several other unknown generators. A somewhat controversial issue appears to be whether activity of the primary auditory cortex contributes to MMN. Study by Javitt et al. (cited by Naatanen et al; 1996) using intracranial recording of MMN to frequency and intensity changes in monkeys indicate primary auditory cortex as MMN generator. In another recent study, Tiitinen et al. (1993) found that the orientation of supratemporal equivalent current dipole for magnetic MMN (MMNm) to frequency change depends on tone frequency. Their results showed that MMNm is generated, at least in part, in a tonotopically organised area of the curved supratemporal auditory cortex. In conclusion, localization of neural mechanisms are involved in auditory sensory memory and involuntary switching of attention (Levanen et al. 1992).

Thus, sites of the generators of any of the late latency potentials are not clearly known. But it can be clearly seen that all these potentials have multiple generators in the cortex.

FACTORS AFFECTING AUDITORY LATE LATENCY RESPONSES

Factors affecting ALLR and MMN summarised as :

(i) Stimulus Characteristics
(ii) Acquisition Characteristics
(iii) Subject Characteristics

The following tabular column summarises the different factors that affect the ALRs and MMN.

(FACTOR	ALR (Exogenous)	ALR (Endogenous) P300	MMN
(A)STIMULUS CHARACTERISTICS (a) Frequency	McCandles(1977) found pure tones better than clicks. Skinner and JoneS (1968) found rise time and fall time of over 20 ms. to be better and more effective in eliciting ALR.	Butcher (1983) reported that changes in the stimulus frequency had no significant effect on P300 response. According to Polich (1989), P300 latency was affected by an interaction of stimulus intensity and duration.	When the physical difference between the standard and the deviant stimuli is small, it is easier for subject to ignore the test stimuli, With a small difference MMN amplitude is low and S/N ratio is poor (Naatanen, 1995). The difference in frequency is more important than the individual frequency of stimuli.
(b) Intensity	The amplitude increases with increase in intensity. The amplitude increaase as a function of intensity were steeper for loer frequency stimuli(500 Hz) than for high frequency (8000 Hz)	Papanicolauetal. (1985) reported, P300 amplitude was not significantly affected by intensity of the stimulus. But study by Butcher (1983) showed that changes in intensity of rare stimulus	Rather than the individual stimulus intensity levels; its the difference intheir intensitylevels, that helps in elicition of MMN. When there is very small difference in the intensity of the two stimuli, the

B.ACQUISITION CHARACTERISTICS **(a) Electrode placement**	Davis, et al. (1939) reported that, response was largest when recorded at vertez is recommended. This was supported by Abe (1954), Codz, et al. (1964), Teas (1965)	P300 is broadly distributed with maximum amplitude observed at midline over the centero-parietalaras. Thus, electrodes should be placee at Fz, Cz and Pz sites for optimum recordings and referenced to linked mastoids (McPherson, 1995)	Again midline electrode placement are found adequate, similar to P300 recording. But Naatanen (1992) recommends use of nose as reference instead of ear or mastoid, because the phase shift in parasaggital and temporal derivations makes it easier to identify the MMN topographically.
(b) Window setting	It is recommended that analysis time should extend for at least 250 msec, post stimulus to include all the ALR peaks (Hall, 1992).	Ritter and Vaughen (1983) found P300 components as late as 450 msec. Hence, the window setting should extend toat least 450-500 msec, post stimulus.	Since peak latency of MMN varies from 80-250 msec, recording window should be at least 300 msec. (Lang, et al. 1995).

	(Antinoro, Skinner and Jones, 1969). It was also found that latency increases as timulus intensities decreases. Also, this isn't entirely linear, since the latency changes is greater for intensity below 45-50 dB SPL (Rapinet al. 1966).	has a significant effect on the latencyof P300 response, hence it is important to test normals and clinical population on same intensity levels.	MMN amplitude is low and S/N ratio is poor (Naatanen, 1995). currently, it is known what the"safe" upper limits are for deviance of intensity. However a positive (P3a) waveform following the MMN, implies that the stimulus difference is too large (cited by Lang et al. 1995).
(c) Interstimulus Inter- ' val **and** Rate on ISI	ALR is highly dependent (Davis et al. 1966; Hari et al. 1982). According to their study, for high intensity levels, ISI, lengthened upto 8 secs, increases the amplitude of ALR waves.	ISI of less than 0.7 secs is recommended by McPherson et al. (1995) while Picton et al. (1981) recommended ISI to be maintained at 1.1 sees. Pictonet al. (1981) reported that as ISI increase the amplitude decreases.	In use of simple stimulus, MMN amplitude increases when ISI is shortened, provided that the intervals between, deviants are of same duration (Naatanen, Paavilainen, Alho, Sams and Reinikainen, 1987). In practise ISI of about 300ms. is recommended for simple stimuli.

(c) Filters	Studies by Ayers et al. (1974); Yamamoto et al. (1979) found frequency composition of ALR in region under 30 Hz. Therefore recommended' filter setting of 1 - 30 Hz/ 100 Hz.	Like other ALR, filter setting of 1-30 Hz. is recommended (Picton, et al.1981).	According to Lang et al. (1995), filter setting of 0.1 to 30 Hz is sufficient for MMN recordings,
(C) SUBJECT CHARACTERISTICS (a) Age	ALR can be recorded from premature, fullterm, new born and older children (Hall, 1992). Studies by Callaway (1975), Goodin et al. (1978) show age related decrease in ALR latency upto 15 years with an increase in latency for persons older thanl5 years.	P300 shows a decrease in latency and an increase in amplitude from age 5-16, followed by progressive decrease in amplitude and increase in latency throughout adulthood (Courchesne, 1978; Goofin, Squires and Henderson, 1978; Polich et al. 1985).	According to Lang et al. (1995), MMN can be without, exception elicited in infants, children and adults and its peak latency shortens with increasing age. A complicated interaction seems to exist between the individual's age and amplitude,

(b) Gender	Onishi and Davis (1968) reported that ALR amplitude in general tended to be larger and amplitude vs. intensity function steeper for females than males.	Most of the s tudies report of no difference among males and females in evoked P300 (Polich et al 1985)- Spongberg and Decker 1985 Fernendez and Torres, 1988).	Gender has an influence on the MMN latency, With complex stimuli; MMN latency is significantly longer in females than in males (Aaltonen et al. 1994).
(c) Attentionand State or arousal	Davis et al. (1964); Picton and Hillyard (1974) found an increase in amplitude with increased stimulus oriented attention, in sleep, latency increased and amplitude decreased (Cody, etal. 1967).	Polich (1986) noted that attention was a necessary factor in th generation of P300 wave and a decrease in attention is related to a decrease in response amplitude.	The use of MMN as an objective measure of auditory functionis to a large extent based on the assumed full or partial indepenance of the MMN from attention, Several studies have shown that the MMN elicited by deviant stimuli, when targets of attended sound sequence or when the sounds are ignored are of very similar amplitude (Naatanen,

Simpson and Loveless, 1982). this suggests that MMN is independent of attention.

In contrast, MMNm (Magnetic MMN) recorded by Aulanko, Hari, et al. (1993) for phonetic change (/ba/ - / da/) was larger in amplitude when attended than when ignored.

Nevertheless,nodata have demonstrated a total disappearance of MMN in absence of attention. Naatanen (1991), therefore suggested that the sensory analysis resulting in neural sound representationis

			not affected,but that the excitability of the MMN process trigered bya deviant stimulus might be dampened in the absence. In summary, it appears that the MMN, to frequency change is strongly attention independent in most conditions.
(d) Effect of drug	Measurement of ALR under sedation is not recommended. Later (1977), observed that use of diazepm resulted in amplitude reductionof N1, P2 and N2 with little effect on latency.	P300 is viewed as active attention condition, and any drug disturbing the alertness of individual would reduce the amplitude of P300 (cited by McPherson, 1996).	In conditions of sleep; small MMN can be seen in stage 2 and REM , phase of sleep (Naatanen et al. 1978). Also MMN could be recorded under pent obarbital anesthesia, (Csepe, et al. 1989), though over limited cortical area and with small amplitude.

LATE LATENCY RESPONSES AND LEARNING DISABLED CHILDREN

Generally, all the investigators have studied interpeak latency, amplitude, lateralization, absolute latency, duration etc, of ALR in LD children. Some also use morphology of waveform. These parameters are compared with the age related normal subjects, as we observe developmental changes in all LLR waveforms.

LATENCY OF THE ALR WAVES IN CHILDREN WITH LEARNING DISABILITY

David et al. (1984) recorded N1, P1, N2 and P2 waves on poor readers and found increased latency of P2 and P1 peaks in poor readers than normal average readers. Satterfield et al. (1984) observed that interpeak latency of P2-P1 peaks were prolonged in learning disabled children. Both these studies used auditory clicks in elicitation of the waves. Increased P2-Pl interpeak latency was attributed to aberrant processing (Satterfleld, 1984). Results of another study by Byring and Jarvileh (1985) using tones, showed that P2 wave occurred at a latency of 170-180 msecs; in poor spellers, while the latency of P2 was found to be around 150 msec, for normal spellers.

Jirsa and Clontz (1989, 1990) recorded N1, P1, N2, P2 and P3 waves using tone pips, in learning disabled children. Their results showed that interpeak latency of P2-P3 was longer than normals in learning disabled. Again another study by Jirsa (1992), using tone burst, found increased P3 latency (700 msec) and reduced amplitude in learning disabled children. This supported the findings of Halgren et al. (1986) and Polich (1986).

Jirsa's (1992) study was supported by Mazzotta and Gallai (1992), who also observed longer P3 latency in dyslexics than normals. In addition, they also reported an asymmetry of P3 scalp distribution at the central and parietal sites in dyslexics, but not in normals.

On the other hand, Segalowitz et al. (1992) did not find any differences between 11 children defined as poor readers and 16 good readers in P3 latencies. Similarly Duncan et al. (1994) did not find any differences in auditory P3 latencies between adult men with severe developmental dyslexia and normally reading men.

But, recently study by Arehole (1995) showed an increased auditory P2-P1, interpeak latency in dyslexics than normals.

Also, Korpilahti and Lang (1991) found differences in MMN component in fourteen learning disabled children and normals. Their results showed that the duration of MMN was significantly smaller in learning disabled than normals, but no differences were found in MMN latency in the two groups.

Thus, it can be seen that no single study has considered all ALRs on same subjects and also study by Segalowitz (1992) and Duncan et al. (1994) did not reveal any difference in P3 latency. Hence, it becomes essential to study all ALRs on same subject and to correlate the findings for conclusive - results.

AMPLITUDE OF ALR WAVES IN LEARNING DISABLED CHILDREN

Byring and Jarvileh (1985) compared the amplitude of P1-N1 waves in poor spellers with normal spellers using tones of 1100 Hz. They found that P1-N1 amplitude was reduced in poor spellers than normals. They suggested that this reduced P1 amplitude could reflect reduced or disturbed early auditory input to left hemisphere in poor readers; whereas the reduced N1 component could be related to processes mediating focussing of attention. Another study by Kibbe et al. (1986) showed delayed and poorly defined N1 in learning disabled than normals. Pinkerton et al. (1989) and Olio and Squires (1989), reported decrease in N1 amplitude in learning disabled than in normals.

Nl differences were found by Neville et al. (1993) in 22 developmental language disordered children, who also had reading disability, when compared to normals. Neville et al. regard their Nl as an equivalent to Nl in adults. An anterior and contralateral distribution of N1, suggests that it represents activity generated in cortex of supratemporal plane; including primary auditory cortex and Heschel's gyri. This was an indication that learning disabled children have auditory temporal processing problems, reduced and slow activity within these brain areas, contributed to their language impairment. The results were taken as support for the hypothesis of a final 'Common path' that arises from an inability to perceive the changing acoustic spectra that characterize the ongoing speech.

Along with N1, PI, P2 and N2; P300 was also studied by different investigators. Holcomb et al. (1986) found smaller P3 amplitudes in three clinical groups including 24 reading disabled. This was interpreted to reflect more active categorical decision making by controls than other groups. Study by Kibbe et al. (1986), also studied P3 amplitude along with other ERPs and found that P3 was absent in learning disabled children.

Similar results of reduced P300 amplitude was also reported by Erez and Pratt (1992) in eleven dyslexics. This was thought to reflect involvement of different cortical structures in detecting relevant target stimuli. Same was reported by Mazzotta and Gallai (1992), with similar interpretation as that of Erez and Pratt (1992). Reduced P3 amplitude was also reported by Pinkerson et al. (1989). Thus, it can be observed that early sensory processing deficits along with other cognitive functional deficits play an important role in learning disability.

MORPHOLOGY : Morphology of the waves has not been studied by many investigators. Jerger and Jerger (1981) reported poor morphology of MLR waves (Middle Latency Responses), but ALR waves were well defined in both ears of LD child.

STUDIES RELATED TO LATERALITY AND HEMISPHERIC ASYMMETRY

Cerebral specialization and hemispheric asymmetry were mostly studied in early 80 's; in learning disabled population, when, processing of speech was thought to be totally specialized in one hemisphere and separated from other processings like music, noise etc.

Early hemispheric differences in ERPs using auditory stimulus, musical cords and voiced words were found by Fried et al. (1981). They compared 8-12 year old normal children with learning disability children; within whom were five dysdeitic children (visual dyslexics) and six dysphonetic children (auditory dyslexics). It was found that; in normals and in dysdeitic children; right hemisphere ERPs were dominant and large showing a clear cut asymmetry inhemispheric processing of stimuli. But the ERPs were found to be same for both words and musical cords in both hemispheres in dysphonetic group showing no asymmetry. This was interpreted to indicate that auditory information processing was not normal in left hemisphere in dysphonetic group. Rosenthal et al. (1982) also found a differential hemispheric asymmetry pattern though not as marked as reported by Fried et al. (1981). Left hemisphere dysfunction in dyslexics has also been reported by Wood et al. (1991), based on his review of studies comparing adults with a history of childhood dyslexics to normally reading adults.

Brunswick and Rippon (1994) also observed similar asymmetry in learning disabled. A deviant laterality index ofNl in dyslexics was observed in their study. These were interpreted to reflect the automatized perceptual processing of phonemic stimuli in view of early sensory ERP effects.

Thus, these studies postulate evidence for left hemisphere dysfunction in dyslexics.

Thus, majority of the studies do show changes in ALR of learning disabled, though few are contradicting. Differences in ALR not only reflect maturational lag, but also a more fundamental processing deficiency (Leppnen and Lyytinen, 1997).

Thus, to detect these fundamental processing deficiencies, in learning disabled, it becomes essential to record all ERPS in same subjects and compare it with age matched normal subjects.

Thus the present investigation studied Nl, PI, N2, P2, P3 and MMN waveforms in learning disabled children.

METHODOLOGY

The present study aimed at studying the following potentials in learning disabled children:

(a) Pl.Nl,P2,N2
(b) P300
(c) MMN

An attempt was made to study

(a) Absolute latency of each wave
(b) Interpeak latency of the wave
(c) N1-P2 amplitude
(d) Duration of MMN

SUBJECTS

A table of 12 subjects, who were diagnosed as learning disabled, using Early Reading Skills Test and Columbia Mental Maturity Scale/Raven's Coloured Progressive Matrices (to find their I.Q) were taken.

The age group was between six to twelve years. Of whom, there were five females and seven males.

These children were confirmed to have peripheral hearing within normal limits using a calibrated audiometer (Madsen OB 822).

Age matched normal subjects served as controls.

INSTRUMENTATION

Madsen OB 822, with TDH-39 earphones lodged in MX-41/AR ear cushions was used for pure tone audiometry, An Electrophysiological Unit, Biologic Auditory Evoked Potentials (Navigator, Systems Corps) was used to record the LLR waves in the subjects.

The picture of the instrument is as shown in Fig. 1.

Accessories :

Electrodes : Five silver chloride disc electrodes were used for recording the potentials (Fig.2).

Earphones : TDH-39 earphones with Mx-41/AR ear cushions, were used to present the stimuli.

TESTING PROCEDURE

Peripheral Hearing Screening

To rule out any peripheral hearing loss, the subjects were screened, using pure tone audiometry. Standard instructions were given to the subject.

Auditory Evoked Potential

- The subject was made to sit comfortably and relaxed.

- Electrode sites were cleaned and electrodes were placed in vertex, parietal, forehead and the two mastoids and connected to electrode box.

- Headphones were placed over the ears of the subject.

- Instructions given were as follows

"There will be two stimuli/tones given to you. One will be frequently occurring one and other is rare one. You have to count the-rare stimuli".

Trial of 15-20 stimuli, where the tester would count along with the subject and familiarize the tones was done. The trials were repeated, untill the subject could count the tones independently.

Electrode **Placement** - Five electrode placement was used.

Position	Connection to Electrode Box
1. Forehead (FPZ)	Common
2. Vertex (Cz)	Channel-1 Input-1
3. Parietal (Pz)	Channel-2 Input -1
4. Mastoid(left)(Al)	Channel-1 Input-2
5. Mastoid (right) (A2)	Channel-2 Input -2

Al and A2 were interlinked using jumper.

After the placement of electrodes, the headphones were placed without disturbing the electrodes.

Before proceeding to start the test, the impedance at electrode sites were kept lower than 5 K ohms and interelectrode impedance lower than 2 Kohms.

STIMULUS PARAMETERS

The following were the stimulus parameters used :

Stimuli	Alternating tone bursts
Frequent stimuli	1000 hz
Infrequent stimuli	2000 Hz
Intensity	70 dB nHL
Filter	0-30 Hz
Repetition Rate	1.1/sec
Rise Time	10 msec
Fall Time	10 msec
Plateau	30 msec
Gain	50,000
Maximum stimuli	300 artifact free stimuli

The test procedure and storing were adopted from the software for LLR (as given in manual).

Analysis:

Latencies and amplitudes of P1, N1, P2 and N2 responses were obtained from the wave of frequent stimuli from Cz wave.

P300 was analysed from the wave of infrequent stimuli (both Cz and Pz waves).

MMN, was obtained from subtraction of the frequent stimuli wave from the wave of infrequent stimuli of the Cz wave.

Latencies of the waves were measured at the peak of each wave. Tf mere was no sharp peak, the centre point was considered and cursor was placed there and latency was measured.

Amplitude of the waves, were measured from the trough to peak and peak to trough of that particular wave.

Duration of MMN, was measured from the onset of negativity to the peak of negativity, called the On-time/On set time and from peak to the end of the response i.e. OFF time.

The waves were measured in Cz/Al-A2 and P2/A1-A2 montage respectively.

Later the stored waves were called and analysed.

Fig. 1 :Biologic Auditory Evoked Potential System (Navigator)

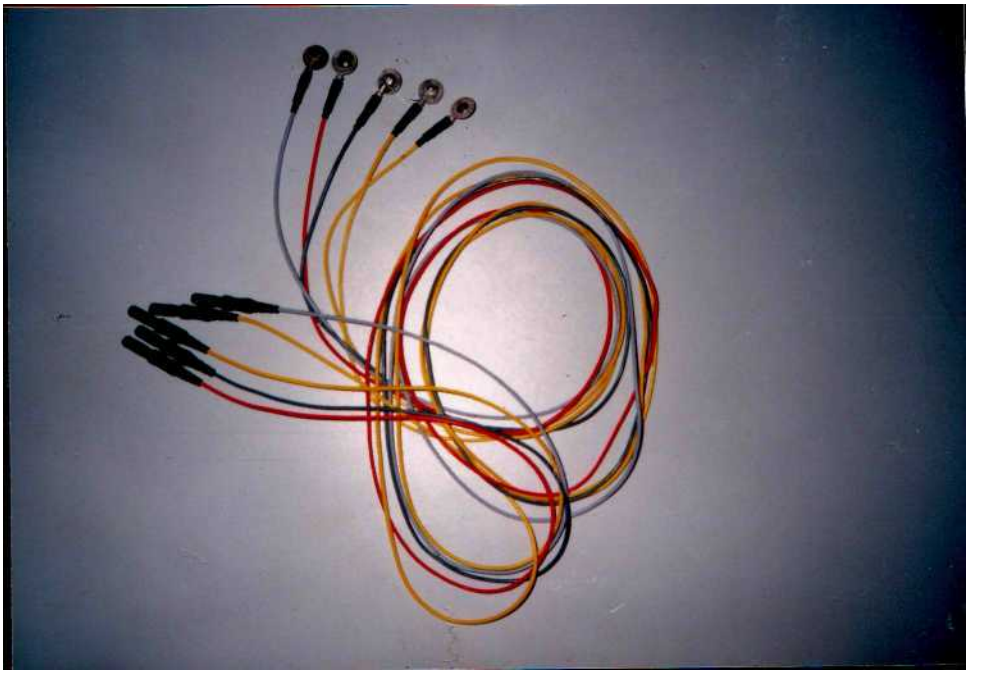

Fig.2: Silver disc electrodes

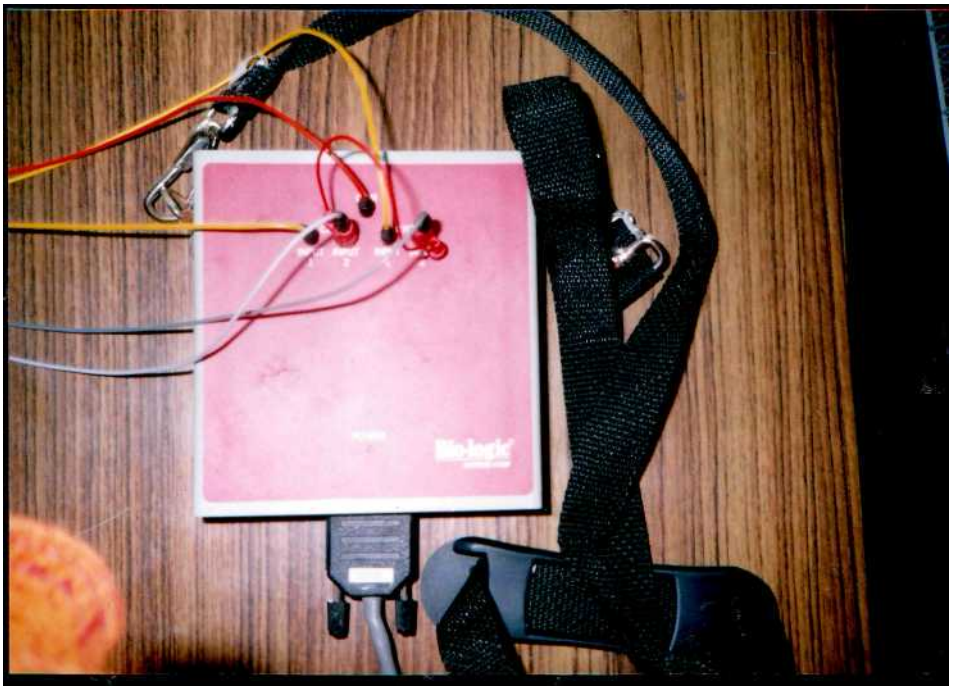

Fig.3 .Electrode box with electrodes.

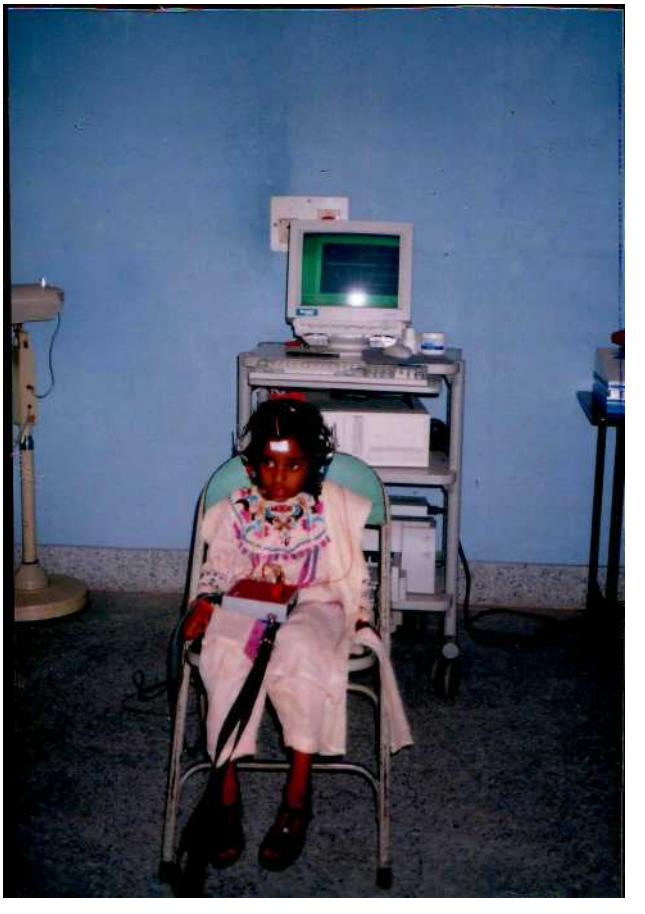

Fig.4 .Subject undergoing testing.

RESULTS AND DISCUSSION

Late Latency Event Related Potentials (both exogenous and endogenous) were recorded in twelve learning disabled children and was compared with age matched normal subjects. Table-1 indicates the number of children in different age groups. Fig.5 shows the normal LLRs and MMN in normal controls.

Table-1 : Number of children in different age groups.

No.of subjects	Age (Years)	Male/Female (M/F)
2	6	1 M and 1 F
4	9	2 M and 2F
5	11	2 M and 3 F
1	14	1F

Analysis of the recorded waveforms revealed the following results.

P1 wave

Table-2 shows the results of latencies of P1 wave in learning disabled subjects.

Table-2 : Latency of P1 wave in learning disabled subjects.

S.No.	Age (Years)	No. of subjects	Latency
1.	6	1	60 msec
2.	9	2	*66* and 69 msec
3.	11	2	61 and 76 msec

Thus P1 could not be identified in seven out of twelve learning disabled (LD) children. The waveforms of the remaining five LD children showed PI wave at a latency which was equivalent to that of their normal

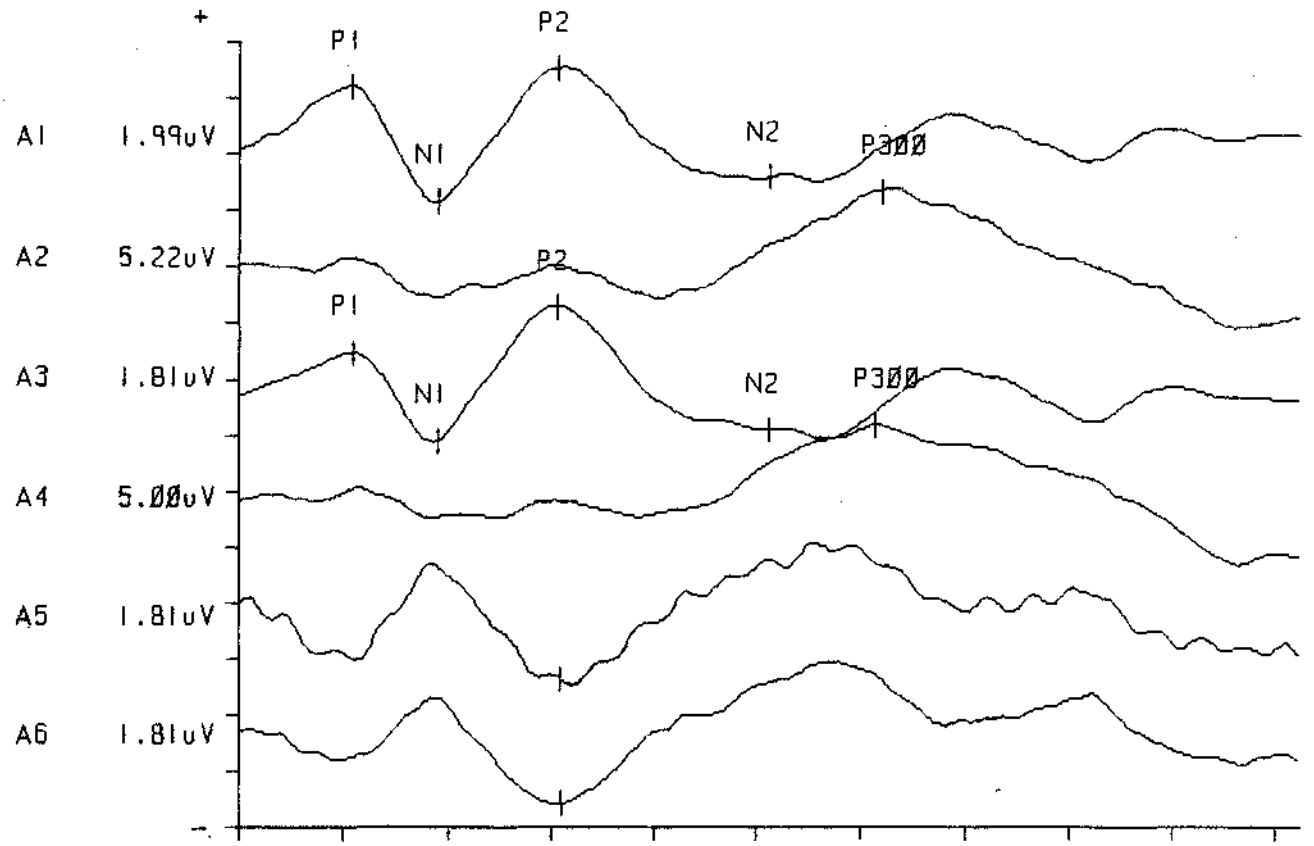

Fig. 5 : Showing normal LLR waves

Al and A2 :Cz waves

Al :Standard stimuli wave

A2 infrequent stimuli wave

A3 and A4 :Pz waves

A5 :A2 -Al - MMN wave

A6 : A4 -A3 - MMN wave

counterparts. The range was within 60-80 msecs. in all the age groups considered in the study. Table-2 shows the latency of PI wave in those children.

This finding is in accordance to the findings of Kibbe et al. (1986), Brying and Jarvileh (1985), who reported of poor P1 responses in Learning Disabled Population.

Though P1 response is considered to be purely exogenous, Hillyard and Picton (1979), say that, P1 may be influenced by some aspect of attention. Most of the earlier studies in literature did not consider P1 response, because it was thought to be a continuation of Middle Latency Response, which has little or minimum effect due to withdrawal of attention to the stimuli. But later on, Hillyard and Picton (1979), explained that attention, may be termed selective (i.e. discriminative), active (i.e. requiring the subject to perform some type of response to the stimuli), passive (not paying attention to the stimuli) and ignore (i.e. requiring the subject to perform some distractive task).

Thus, when any of these are affected, though subtly, it would affect the response. In this study, with seven out of twelve LDs not having an identifiable P1 and the remaining showing poor P1 response, it can be **concluded** that, attention necessarily plays a role in generation of P1 and **when** it is deviant, along with other waves, it can be concluded as a condition depicting auditory processing problem. This is also supported by the studies of generation sites of P1, which traces its origin to late thalamic projections, which controls most of the attention processes. Thus it becomes important to consider the P1 response also, for diagnosis of auditory processing problem.

Nl Waves

In this study it was found that, Nl occurred within the latency of 80 msec, to 110 msec, with an amplitude of 6.90 /uV to 8.32 /uV in normal subjects.

Table-3 : Latency and amplitude of Nl wave in Learning Disabled.

Sl.No.	Age	No.of subjects	Latency (msec)	Amplitude (uV)
1.	6	1	153	0.90
2.	9	2	114 & 133	1.04,1.32
3.	11	2	113 & 123	1.65,1.93

Nl could not be identified in the same seven subjects in whom PI also, could not be identified. In the remaining five LDs, the latencies were prolonged as shown in Table-3 and their amplitudes were reduced. Fig. 6 shows the absence of PI, Nl and P2 responses in LD children.

These findings support the results obtained by David et al. (1985), Kibbe et al. (1986), Pinkerton et al. (1989) and Olio and Squires (1989). These studies also report of increased latency and decreased amplitude of Nl responses in LD children, when compared to the normal subjects. Nl deviancy was also reported by Neville et al. (1983), in children with reading - disability, who explained this as an indication that LD children have auditory temporal processing problems, that indicates reduced and slow activity within supratemporal plane, including primary auditory cortex and Heschel's gyri.

According to Picton and Hillyard (1974), Nl responses was related to state of arousal and the attention processes. They reported that Nl component shows an increase in amplitude of approximately 0.61 uV when the stimulus is attended. Also, Naatanen and Picton (1987) have suggested that, somewhere around six different processing activities occur within the time period between the P60 and P160 (i.e. P1 and P2), including the mismatched negativity and a processing negativity. Thus, it is thought that, the changesdue to attention observed in the Pl-Nl may actually reflect a negativity that begins shortly after stimulus onset and peaks at approximately 100 msecs. Thus, when the attention process is absent, there is no peaking of negativity and thus, it is seen that no Nl is found

during unattended conditions or if it is present, it is of lower amplitude. Also, the increase in latency of N1, seen in this study, suggests that, the LD children take longer time to initiate the negativity, i.e. in turn to initiate the attention process itself on whole. Thus both latency and amplitude of Nl wave along with findings of other waves, should be considered in diagnosing auditory processing problems. It can be concluded that reduced amplitude of Nl and its increased latency, almost always conveys a problem in cognitive processing of the auditory stimulus and thus confirms the auditory processing problem, along with deviancy in other waves also.

P2 Wave

Table-4 : Latencies of P2 wave in learning disabled children

Sl.No.	Latency	Total No. of subjects	Age (in years)		Pl/Nl condition
			No.	Age	
1.	Could not be identified	8	3	11	No P1 and - Nl
			2	9	
			1	6	
			1	14	
			1	9	P1 andNl present (Nl prolonged)
2.	169-184 msec (prolonged)	2	1	9	P1 andNl present (Nl prolonged)
			1	11	
3.	120-150 (normal range)	2	1	11	P1 and Nl present Nl (prolonged)
			1	6	

Thus as shown in Table-4 eight out of twelve LDs showed no P2 response, while in the remaining there was identifiable P2. In the remaining four, two had normal latency (120-150 msec) and the other two had prolonged latency (169-184 msec). The N1-P2 amplitude was reduced in all the four LD cases; in whom P2 could be identified. Fig. 6 shows absence of P1, Nl, P2 waves in LD children.

Similar results of increased latency of P2, was reported by David et al. (1984), Byring and Jarvileh (1985). The latencies found in this study (i.e. prolonged P2) are in accordance with those found by Byring and Jarvileh (1985) (170-180 msec), in learning disabled, while in normals it was around 150 msec.

The role of attention in latency and amplitude of P2 was proved by studies of James et al. (1989), Woods et al. (1992) for pure tones. Also, the P2 response changes somewhat as the stimuli becomes more complex eg. in cognitive science it is thought that information processing appear to be dependent on selective attention, which is referred to as controlled processing and information processing that is not dependent on selective attention, is referred to as automatic processing. It can be considered that the P1 and Nl are dependent on both automatic and controlled processing, with more emphasis on automatic processing, but still dependent on controlled processing also. Similarly P2 in a response that is dependent on both, with emphasis on controlled processing man automatic processing (Ritter et al. 1983).

Hence, it can be seen that, any deficit in controlled attention also affects P2 wave and reduces its amplitude. Not many studies have reported on latency of P2, probably because of the wide range of latency seen in normals itself than P1 and Nl. Thus, any deviancy in P2 wave should be considered in diagnosis of auditory processing of temporal aspects of stimuli, which requires more controlled attention.

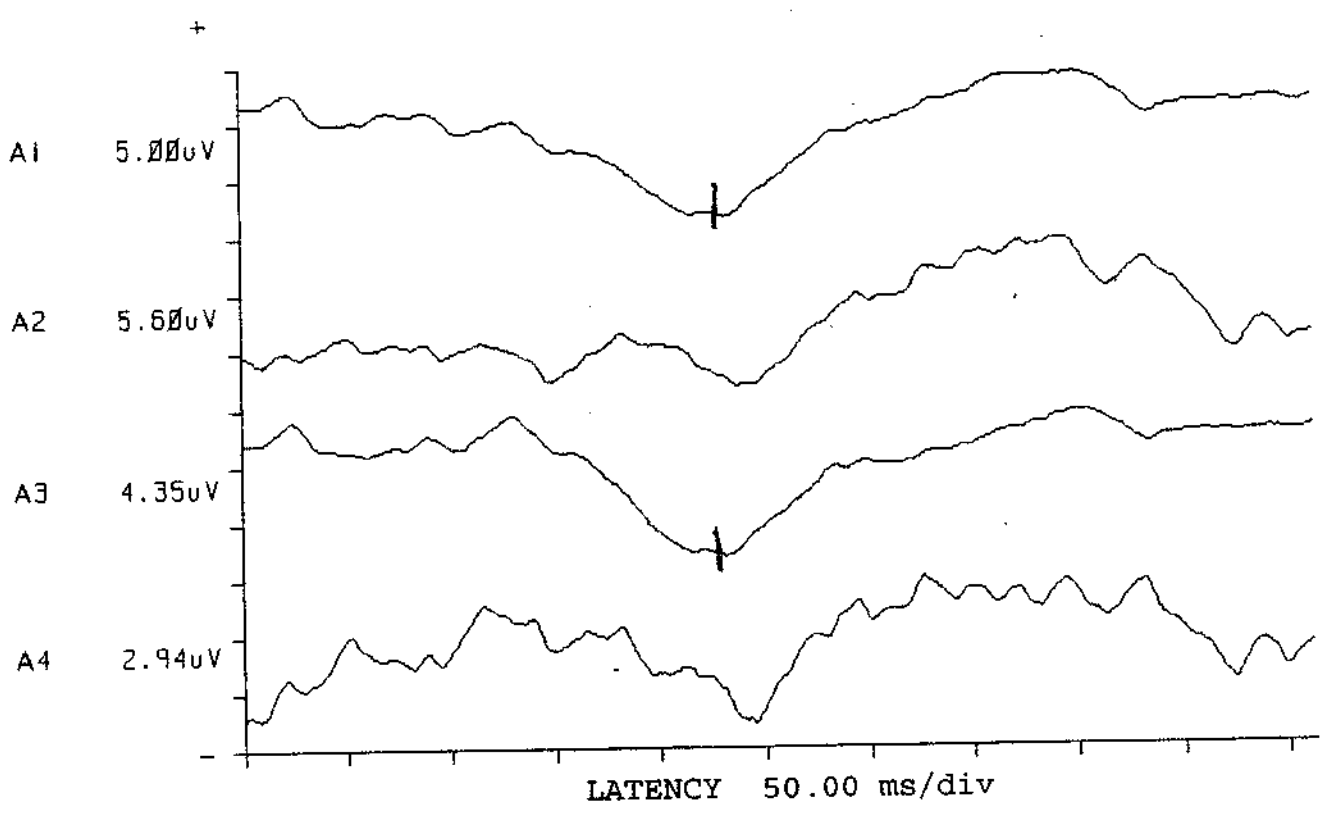

Fig. 6 : Showing absence of P1, N1 and P2
A1 and A2 : Cz waves
A1 : Standard stimuli wave
A2 : Infrequent stimuli wave

N2 wave

It is seen that out of twelve LDs, only two did not show any N2, while in the remaining ten of them, there was identifiable N2. Also, the latencies were same as that of their normal controls (177-230 msec).

According to Ritter et al. (1983) N2 response, along with P2, changes as the stimuli become more complex. This N2, is found to more affected by the controlled attention than automatic. Ritter et al. (1983), demonstrated that N2 had equal amplitude in both attended and unattended condition for complex stimuli, or a stimuli where decision has to be made. According to their study using visual stimuli, N2 represented two stages of processing. That is, N2 is influenced by the physical dimensions of stimuli related to automatic processing and also semantic dimensions of stimuli related to controlled processing. It is strongly indicative of N2 being capable of classifying the stimulus. According to them, "... *our interpretation of these results is that the N2, is a generic component, which reflects a stage of processing (stimulus classification) which occurs whether the stimulus being classified are tones, vertical or horizontal bars or words".*

Thus, we know that, in speech perception, in perception of consonants, it is categorical perception, that helps us to discriminate one consonant from other. This ability to categorise as sounds belonging to one category is very important to perception of speech. Thus any deviancy in this wave (N2) is indicative of inability to classify the stimulus. The absence of wave becomes more informative than its presence. If the wave is absent, or is of low amplitude, then it is considered that categorization of stimuli is affected.

P300 wave

Analysis of the waves showed that, in nine of the twelve LDs P300 could not be identified. Out of the remaining 3, one of them had Bifid P300. The latencies of these three LDs P300, were found to be within normal range (265 msec to 335 msec). However, the amplitudes of these

three LDs were reduced to 1,55 uV to 2.93 uV, while in normals it was around 3.30 uV to 6.9 uV. Fig.7 shows absence of P300 in LD children.

Out of these three, one was fourteen year old female, other were two, eleven year old male subjects. P1 N1, P2 was not identified in the fourteen year old female LD and in one of the eleven year old male.

While, in only one; eleven year old LD (male) all the waves could be identified.

Thre was no difference in the Cz and Pz waves in latencies and amplitude of P300. Both were found to be similar.

A point to be noted is that, P300 was present only in the two eleven year old children and one fourteen year old LD, while the six and nine years old has no P300 response. This probably shows a developmental lag in the development of attention and cognitive skills, because, as the age increased P300 waves were identifiable, and also the amplitude of fourteen year old was similar to that of eight-nine year old normal subjects and that of eleven year old was similar to six-seven year old normal subject.

This result of no P300 and reduced amplitude ofP300, is in accordance to the results obtained by Jirsa and Clontz (1990), Jirsa (1992), Halgren, et al. (1986), Polich (1986), Mazzotta and Gallai (1992), Holcomb/et al. (1986), Kibbe et al. (1986), Erez and Prett (1992) and Pinkerton et al. (1989). According to Pinkerton et al. (1989) absence of P300 is an indication of early sensory processing deficits along with other cognitive functional deficits in learning disabled children.

It has been suggested that the P300 is associated with controlled processing since the amplitude of the response is larger for a stimulus in the attended condition than in the ignore condition. According to Picton and Hillyard (1979), the P300 may be viewed in essentially three attention conditions, (i) active, (2) passive and (3) ignore conditions. P300 is found to be most robust in active attending condition. It can be seen from their

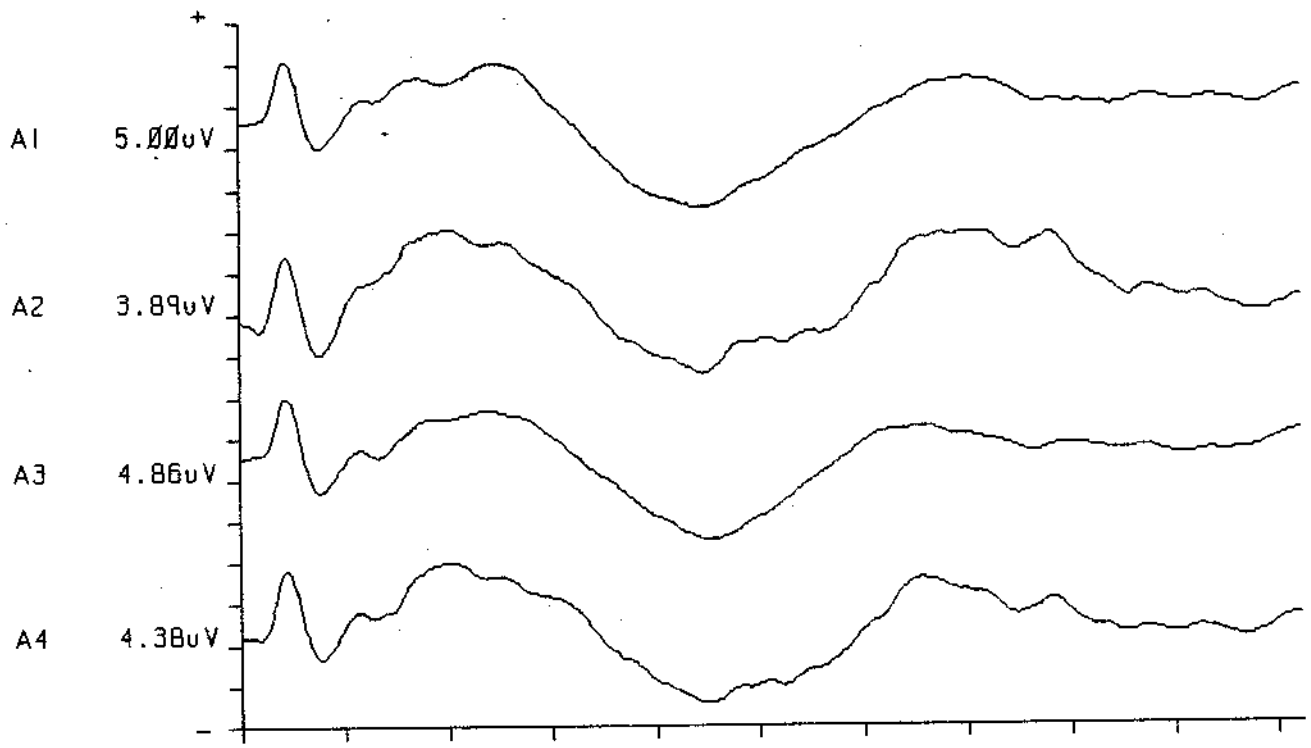

Fig.7 :Showing absence of P300 wave in LD case
A2 :CZ infrequent stimuli
A4 :Pz infrequent stimuli

study, that, the components peaking between 80 msec, to 100 msec, show a greater amplitude in the attended condition than in an unattended and those peaking between 250-400 msec, are enhanced when a target stimuli is recognized. Hillyard,Hink, Schwentand Picton (1973), propose thatNl amplitude i.e. *"indexes the stimulus set which selectively excludes sensory input to the unattended",* condition from being processed, whereas the P300 reflects, *"selective recognition which is coupled with an appropriate cognitive response".* Hence as it is a more direct reflector of cognitive processes, its absence and presence both are significant in diagnosis of auditory processing problems.

As a measure, interpeak latency of P2-P3 could not be considered in this study because, both the peaks were present only in two cases and the latencies were found to be with in normal range. Though there are studies by Kibbe et al. (1986), who say of prolonged P2-P3 latency, it is seen that the P300 in their was recorded at 700 msec, in normals itself. Thus, interpeak latency could not be taken as an indicator of auditory processing problem in the present study.

Morphology of the Waveform

Eleven out of twelve LD children has poor morphology of almost all waves. Only one of them had good morphology. This is contradictory to the findings of Jerger and Jerger (1981), who report of good morphology of LLR waves in both ears of LD child. But, the poor morphology can be accepted due to its consistency seen in almost all LDs studied. Also, this poor morphology of wave reflects a cleared maturational lag of the supertemporal region, reticular formation and the hippocampus region, Heschle's gyrus and association areas, which are found to be the major generator sites of LLR waves.

MMN wave

Along with all these LLR waves mismatch negativity (MMN) was also recorded and analysed. According to Naatanen and Picton (1987),

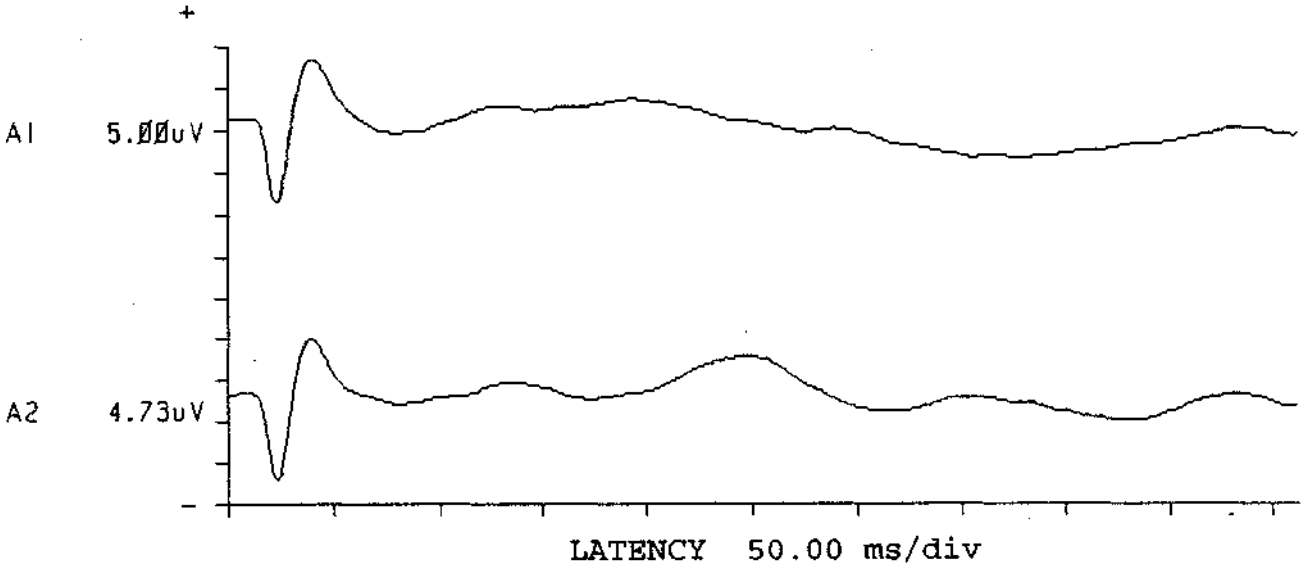

Fig. 8 : Showing absence of MMN in LD case

A1 : Cz wave

A2. :Pz wave

(Only Cz waves were considered)

there are six different processing activities occur within the period between P60 and P160 and MMN represents one or more of these. MMN was absent in three out of 12 LDs. The latencies of the remaining nine were similar to those of their normal counterparts (100-150 msec). In duration of MMN, both rise time and off time was measured in LD and normals. In normals, both rise time and off time were almost of same duration, but in LDs five out of nine had no off times. Also the duration of MMN in LDs was found to be drastically reduced to 26-50 msec, while it was 60-75 msec, in normals. Fig. 8 shows absence of MMN in LD child.

This reduction shows that the attention process is incomplete and does not reach the peak negativity showing complete processing activity. MMN, thought to be measuring subliminal attention, its reduction show low or no subliminal attention. This results in absence and low duration of MMN is supported by study of Korpilatiti and Lang (1991). Amplitude measures in MMN is not considered, because of their high variability even in normals.

General morphology revealed a clear reduction in amplitude of MMN in LDs. Normals showed an amplitude of 5-7.84 uV while it was only 0.91 to 3.89 uV in LDs.

Thus, by this analysis, it is found that LLR waves are clear indication of the presence of auditory processing problems. The reduction is amplitude and increase in latency of these waves, are highly conclusive of the presence of auditory processing problems in learning disabled children.

SUMMARY AND CONCLUSION

Central Auditory Processing problems in learning disabled children has been studied for long using behavioural tests. With the advent of electrophysiological tests this task of testing CAPD has become more objective and easier in testing for children with low speech and language skills.

This study was undertaken to see if the electrophysiological tests like, Auditory Late Latency Potentials, revealed any auditory processing problems in learning disabled children.

The study aimed at recording the auditory LLRs including P300 and MMN in LD children and age matched normal subjects.

These potentials were recorded in twelve learning disabled children and in age matched normals using Biologic Auditory Evoked Potential System (Navigator). These children were confirmed to have hearing within normal limits using pure tone audiometry, before recording the potentials.

Analysis of the results show that almost all learning disabled had deficits in the waveforms. The abnormalities seen included the following:

a. Absence o fPl, N1,P2,N2, P3 and/or MMN.
b. Prolonged latency of P2, Nl and P3.
c. Reduced amplitude of all the waves.
d. Poor morphology of the waves.
e. Reduced duration of MMN wave.

A majority of the LD children showed poor morphology of all waves. Both exogenous and endogenous potentials were affected in nearly eleven out of twelve learning disabled children. In one subject, all the waves except MMN, were found to be normal.

Thus, the results of this study suggest that if all the LLRs or if at least any of the LLRs are seen to be affected in terms of reduced amplitude, prolonged latency or poor morphology or of reduced duration as in case of MMN, auditory processing problem can be suspected. As this test is not language specific, it can be used in any linguistic population. In other words it can be concluded that LLRs (both exogenous and endogenous) are good tools to diagnose auditory temporal processing problems in learning disabled population. Also, based on the results, the hypothesis that learning disabled children have auditory processing problems, is confirmed and accepted.

Limitations of the Study

1. Only a small group of population was included for thestudy.

2. As it was a heterogeneous group, no statistical measurescould be applied.

3. Multiple electrode placement, could have given betterresults in terms of morphology of waves.

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